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☐	L60	(shek)adj(theresa)	3
☐	L59	L58 and (EG-VEGF)	3
☐	L58	(wood)adj(william)adj(i)	2330
☐	L57	L56 and EG-VEGF	3
☐	L56	(watanabe)adj(colin)	1760
☐	L55	L54 and EG-VEGF	0
☐	L54	L53 and antibod?	88
☐	L53	l48 and PRO1186	88
☐	L52	L48 and 1186	90
☐	L51	L49 and EG-VEGF	7
☐	L50	L49 ane EG-VEGF	58117
☐	L49	L48 and antibod?	468
☐	L48	(ferrara)adj(napoleone)	513
☐	L47	L42 and (TANGO)adj(266)	0
☐	L46	L42 and TANGO266	0
☐	L45	L42 and prokineticin	1
☐	L44	L42 and PRO1186	12
☐	L43	L42 and EG-VEGF	0
☐	L42	530/387.1, 387.3, 388.1, 388.15, 388.23.ccls.	6927
☐	L41	L34 and (prokineticin)adj(1)	0
☐	L40	L34 and (human)adj(TANGO)adj(266)	0
☐	L39	L34 and (human)adj(TANGO266)	0
☐	L38	L34 and (TANGO)adj(266)	0
☐	L37	L34 and zven	1
☐	L36	L34 and PRO1186	2
☐	L35	L34 and EG-VEGF	0
☐	L34	424/130.1, 133.1, 135.1, 139.1, 141.1, 142.1, 145.1.ccls.	17686
☐	L33	PTA-4122	0
☐	L32	PTA-4121	0
☐	L31	PTA-4120	0

☐	L30	PTA-4119	0
☐	L29	L28 and PTA-4119	0
☐	L28	L27 and antibod?	33
☐	L27	EG-VEGF	45
☐	L26	L25 and anti-EG-VEGF	0
☐	L25	L13 and pro1186	669
☐	L24	L23 and EG-VEGF	0
☐	L23	L22 and VEGF	578
☐	L22	L21 and antibody	692
☐	L21	L15 and pro1186	692
☐	L20	L19 and EG-VEGF	0
☐	L19	L18 and VEGF	88
☐	L18	L17 and antibody	88
☐	L17	L11 and pro1186	88
☐	L16	L15 and EG-VEGF	3
☐	L15	(wood)adj(william)adj(I)	2330
☐	L14	L13 and EG-VEGF	3
☐	L13	(watanabe)adj(colin)	1760
☐	L12	L11 and EG-VEGF	9
☐	L11	(ferrara)adj(napoleone)	513
	DB=USPT; PLUR=YES; OP=OR		
☐	L10	L9 and EG-VEGF	0
☐	L9	(530/387.1).ccls.	2184
☐	L8	L7 and PTA-4122	0
☐	L7	L6 and PTA-4121	0
☐	L6	L3 and PTA-4120	0
☐	L5	L3 and PTA-4119	0
☐	L4	L3 and PRO1186	0
☐	L3	L1 and VEGF	136
☐	L2	L1 and EG-VEGF	0
☐	L1	(424/130.1 |424/131.1 |424/132.1 |424/133.1 |424/134.1 |424/135.1 |424/136.1 |424/137.1 |424/138.1 |424/139.1 |424/140.1 |424/141.1).ccls.	2452

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=> s EG-VEGF
L1 166 EG-VEGF

=> s l1 and antibod?
L2 13 L1 AND ANTIBOD?

=> dup remove l2
PROCESSING COMPLETED FOR L2
L3 9 DUP REMOVE L2 (4 DUPLICATES REMOVED)

=> d l3 1-9 cbib abs

L3 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2005:902707 Document No. 143:246742 Chimeric immunogens employing VEGF and helper T-cell peptide epitopes. Kaumaya, Pravin; Cohn, David (The Ohio State University Research Foundation, USA). PCT Int. Appl. WO 2005076972 A2 20050825, 65 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2005-US3747 20050207. PRIORITY: US 2004-2004/PV542041 20040205.
AB The authors disclose compns. and methods of treating patients with malignancies associated with overexpression of VEGF, particularly ovarian cancer. In one example, the compns. comprise VEGF epitopes and chimeric peptides comprising one or more of said epitopes and a T cell epitope.

L3 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2005:523320 Document No. 143:53487 Treatment of rheumatoid arthritis with hypoxia-inducible factor 1α antagonists. Defranoux, Nadine; Hurez, Vincent Jacques; Michelson, Seth G.; Shoda, Lisl Katharine; Wennerberg, Leif Gustaf (Entelos, Inc., USA). PCT Int. Appl. WO 2005053744 A1 20050616, 72 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA,

BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-US39484 20041124. PRIORITY: US 2003-525363P 20031126.

AB The invention encompasses a novel method of treating inflammatory disease, such as rheumatoid arthritis, and novel methods of identifying and screening for drugs useful in the treatment of inflammatory diseases and their clin. symptoms. The inventors have made the discovery that the activity of HIF-1α, a transcription regulator known to have an effect on some cancers, has a significant impact on the pathophysiol. of rheumatoid arthritis. The symptoms of an inflammatory disease, such as rheumatoid arthritis, may be alleviated by administering a compound that inhibits the activity of HIF-1α.

L3 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2004:780858 Document No. 141:273369 Compositions comprising human and mouse Bv8 and EG-VEGF with hematopoietic and immune activity and therapeutic uses for blood diseases and immune disorders. Ferrara, Napoleone; Lecouter, Jennifer (Genentech, Inc., USA). PCT Int. Appl. WO 2004081229 A2 20040923, 161 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-US7622 20040312. PRIORITY: US 2003-454462P 20030312; US 2003-511390P 20031014.

AB The present invention relates to the novel expression and activities of Bv8 and EG-VEGF in hematopoietic stem cells (HSCs), lineage-committed blood progenitor cells, and lymphocytes. In particular, Bv8, EG-VEGF, and their receptors are expressed in bone marrow HSCs, peripheral blood leukocytes (PBLs), as well as many hematol. malignant cell lines. Bv8 and EG-VEGF are capable of promoting colony formation of bone marrow mononuclear cells and spleen-derived progenitor cells, increasing populations of white blood cells, and promoting activation of B lymphocytes and T lymphocytes. BvS8 nucleic acids and polypeptides, EG-VEGF nucleic acids and polypeptides, or combinations thereof can be used in a number of assays and in diagnosis and treatment of conditions associated with hematopoiesis, neutropenias, immunodeficiency disorders, and autoimmune disorders.

L3 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2004:75976 Document No. 140:139512 EG-VEGF receptor antagonists, therapeutic and diagnostic methods, and antagonist identification test system. Haendler, Bernard; Hess-Stumpp, Holger; Schmidt, Anja (Schering A.-G., Germany). Ger. Offen. DE 10229379 A1 20040129, 13 pp. (German). CODEN: GWXXBX. APPLICATION: DE 2002-10229379 20020626.

AB The invention discloses the pharmaceutical use of inhibitors of EG -VEGF-polypeptide and EG-VEGF-nucleic acid and/or the corresponding receptors for treatment and diagnosis of endometriosis and endometrial carcinoma and for treatment of dysfunctional bleeding. The invention further discloses the use of EG-VEGF analogs to increase fertility rates. The invention also discloses a test system for identification of EG-VEGF receptor antagonists.

L3 ANSWER 5 OF 9 MEDLINE on STN DUPLICATE 1

2004388609. PubMed ID: 15292351. Human endocrine gland-derived vascular endothelial growth factor: expression early in development and in Leydig cell tumors suggests roles in normal and pathological testis angiogenesis. Samson Michel; Peale Franklin V Jr; Frantz Gretchen; Rioux-Leclercq Nathalie; Rajpert-De Meyts Ewa; Ferrara Napoleone. (Department of Molecular Oncology, Genentech, Inc., South San Francisco, California 94080, USA.) The Journal of clinical endocrinology and metabolism, (2004 Aug) Vol. 89, No. 8, pp. 4078-88. Journal code: 0375362. ISSN: 0021-972X. Pub. country: United States. Language: English.

AB Angiogenesis is essential for tumor growth and metastasis. A new human angiogenic mitogen, endocrine gland-derived vascular endothelial growth factor (EG-VEGF), has been recently identified; its expression pattern is restricted to endocrine glands, with the highest expression in testis. We used in situ hybridization and newly generated monoclonal antibodies to investigate the expression of EG-VEGF in normal human prenatal and adult testis and in 48 human testicular tumors of different subtypes. We found that EG-VEGF was expressed from 14 wk until birth in human fetal testis. In the adult testis, EG-VEGF was strongly expressed only in Leydig cells. In testicular tumors, EG-VEGF was expressed specifically in Leydig cell tumors, whereas germ cell-derived neoplasms, including carcinoma in situ, seminoma, and nonseminomatous germ cell tumors, were negative for this antigen. In contrast, VEGF, another powerful angiogenic factor, was expressed in seminoma, but very weakly in Leydig cell tumors. Interestingly, we found that Leydig cell tumors presented vessel surface density 3.2-fold higher than seminoma. These findings argue that human EG-VEGF may play a role in angiogenesis both during the early endocrine development of testis and in the adult testis as well as in Leydig cell tumor growth.

L3 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2003:633928 Document No. 139:175555 Drug screening for inhibitors of peptide ligands for G-protein-coupled receptors ZAQ and 15E as angiogenesis inhibitors. Ohtaki, Tetsuya; Masuda, Yasushi; Takatsu, Yoshihiro (Takeda Chemical Industries, Ltd., Japan). PCT Int. Appl. WO 2003066860 A1 20030814, 308 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (Japanese). CODEN: PIXXD2. APPLICATION: WO 2003-JP1057 20030203. PRIORITY: JP 2002-27299 20020204.

AB Provided are a method and kit for screening compds. inhibiting the activity of novel peptide ligands for two orphan G-protein-coupled receptors ZAQ and 15E. Such compds., antisense nucleic acids or antibodies, are usable as, for example, angiogenesis inhibitors in diagnosis, prevention, and therapy for cancer, polycystic ovary syndrome, ovary overstimulation, etc. The amino acid sequences of those peptides, human, mouse, rat, and bovine ZAQ ligand peptide, snake venom MITI and human and other mammalian homolog (Bv8 peptide), are provided. Endocrine gland-derived vascular endothelial growth factor (EG-VEGF, identical to prokineticin 1) is a novel peptide recently identified as a selective mitogen for endocrine gland endothelial cells. The present study demonstrates that EG-VEGF/prokineticin 1 and a peptide closely related to EG-VEGF, prokineticin 2, are cognate ligands of two orphan G-protein-coupled receptors designated ZAQ (= EG-VEGF/PK-R1) and 15E (= EG-VEGF/PK-R2). EG-VEGF/prokineticin 1 and prokineticin 2 induced a transient increase in intracellular calcium ion concentration ([Ca2+]i) with nanomolar potency in Chinese hamster ovary (CHO) cells expressing EG-VEGF/PK-R1 and -R2 and bind to these cells with high affinity and with

different receptor selectivity. EG-VEGF/prokineticins provoke rapid phosphorylation of p44/42 MAP kinase and DNA synthesis in the bovine adrenal capillary endothelial cells (BACE). The mRNAs of both EG-VEGF/PK-R1 and -R2 were expressed in BACE. The identification of the receptors for EG-VEGF /prokineticins may provide a novel mol. basis for the regulation of angiogenesis in endocrine glands.

L3 ANSWER 7 OF 9 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

2003:439594 The Genuine Article (R) Number: 682FM. Differential expression of the angiogenic factor genes vascular endothelial growth factor (VEGF) and endocrine gland-derived VEGF in normal and polycystic human ovaries. Ferrara N (Reprint); Frantz G; LeCouter J; Dillard-Telm L; Pham T; Draksharapu A; Giordano T; Peale F. Genentech Inc, Dept Mol Oncol, 1 DNA Way, San Francisco, CA 94080 USA (Reprint); Genentech Inc, Dept Mol Oncol, San Francisco, CA 94080 USA; Genentech Inc, Dept Pathol, San Francisco, CA 94080 USA; Univ Michigan, Dept Pathol, Ann Arbor, MI 48109 USA. AMERICAN JOURNAL OF PATHOLOGY (JUN 2003) Vol. 162, No. 6, pp. 1881-1893. ISSN: 0002-9440. Publisher: AMER SOC INVESTIGATIVE PATHOLOGY, INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3993 USA. Language: English. *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Angiogenesis is a key aspect of the dynamic changes occurring during the normal ovarian cycle. Hyperplasia and hypervascularity of the ovarian theca interna and stroma are also prominent features of the polycystic ovary syndrome (PCOS), a leading cause of infertility. Compelling evidence indicated that vascular endothelial growth factor (VEGF) is a key mediator of the cyclical corpus luteum angiogenesis. However, the nature of the factor(s) that mediate angiogenesis in PCOS is less clearly understood. Endocrine gland-derived (EG)-VEGF has been recently identified as an endothelial cell mitogen with selectivity for the endothelium of steroidogenic glands and is expressed in normal human ovaries. in the present study, we compared the expression of EG-VEGF and VEGF mRNA in a series of 13 human PCOS and 13 normal ovary specimens by in situ hybridization. EG-VEGF expression in normal ovaries is dynamic and generally complementary to VEGF expression in both follicles and corpora lutea. A particularly high expression of EG-VEGF was detected in the Leydig-like hilus cells found in the highly vascularized ovarian hilus. in PCOS ovaries, we found strong expression of EG-VEGF mRNA in theca interna and stroma in most of the specimens examined, thus spatially related to the new blood vessels. In contrast, VEGF mRNA expression was most consistently associated with the granulosa cell layer and sometimes the theca, but rarely with the stroma. These findings indicate that both EG-VEGF and VEGF are expressed in PCOS ovaries, but in different cell types at different stages of differentiation, thus suggesting complementary functions for the two factors in angiogenesis and possibly cyst formation.

L3 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2002:10528 Document No. 136:65270 Protein and cDNA sequences encoding human EG-VEGF protein and methods of use. Ferrara, Napoleone; Watanabe, Colin; Wood, William I. (Genentech, Inc., USA). PCT Int. Appl. WO 2002000711 A2 20020103, 133 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US20116 20010622. PRIORITY: US 2000-213637P 20000623; US 2000-230978P 20000907; WO 2000-US32678 20001201.

AB The present invention is directed to novel polypeptides designated herein

as EG-VEGF of human and to nucleic acid mols. encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Also provided herein are methods of screening for modulators of EG-VEGF. Furthermore, methods and related methods of treatment are described herein which pertain to regulating cellular proliferation and chemotaxis.

L3 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2002:964996 Document No. 138:33697 Endocrine gland-derived vascular endothelial growth factor nucleic acids and polypeptides and their biological activities and use in drug screening and therapies. Ferrara, Napoleone; Watanabe, Colin; Wood, William I.; Shek, Theresa (USA). U.S. Pat. Appl. Publ. US 2002192634 A1 20021219, 105 pp., Cont.-in-part of U.S. Ser. No. 886,242. (English). CODEN: USXXCO. APPLICATION: US 2001-27603 20011219. PRIORITY: US 1998-96146P 19980811; WO 1999-US12252 19990602; US 1999-145698P 19990726; US 1999-380137 19990825; WO 2000-US219 20000105; WO 2000-US4914 20000224; WO 2000-US8439 20000330; US 2000-213637P 20000623; US 2000-230978P 20000907; US 2000-709238 20001108; WO 2000-US32678 20001201; US 2001-886242 20010620.
AB The present invention is based on the identification and characterization of a novel, tissue-restricted, growth and differentiation factor that acts selectively on one endothelial cell type. This factor, referred to as endocrine gland-derived vascular endothelial growth factor (EG-VEGF), induces proliferation, migration, and fenestrations in capillary endothelial cells derived from endocrine glands, but has no effect on a variety of other endothelial and non-endothelial cell types tested. EG-VEGF also induces phosphorylation of kinases involved in cell proliferation or survival, including ERK1, ERK2, Akt, and eNOS. EG-VEGF nucleic acids and polypeptides can be used in a number of assays and in diagnosis and treatment of conditions associated with hormone-producing tissue. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Also provided herein are methods of screening for modulators of EG-VEGF. Furthermore, methods and related methods of treatment are described herein which pertain to regulating cellular proliferation and chemotaxis.

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L4 5 "PRO1186"

=> s l4 and antibod?
L5 3 L4 AND ANTIBOD?

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L6 3 DUP REMOVE L5 (0 DUPLICATES REMOVED)

=> d l6 1-3 cbib abs

L6 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
2002:773696 Document No. 137:289998 Cloning, protein and cDNA sequence of human protein PRO1186. Baker, Kevin; Chen, Jian; Goddard, Audrey; Gurney, Austin L.; Smith, Victoria; Watanabe, Colin K.; Wood, William I.; Yuan, Jean (Genentech, Inc., USA). Eur. Pat. Appl. EP 1247863 A1 20021009, 49 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL. (English). CODEN: EPXXDW. APPLICATION: EP 2002-12968 19990602.

PRIORITY: US 1998-96146P 19980811; EP 1999-955293 19990602.

AB The invention relates to protein and cDNA sequence of human protein PRO1186. The invention relates to methods and vector for recombinant expression of protein PRO1186 in mammalian cell, yeast, Escherichia coli and insect cell. The invention relates to antibody against protein PRO1186 and drug screening.

L6 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

2001:417147 Document No. 135:29838 Secreted and transmembrane proteins identified by sequence comparison and cDNAs encoding them and their uses. Baker, Kevin; Beresini, Maureen; Deforge, Laura; Desnoyers, Luc; Filvaroff, Ellen; Gao, Wei Qiang; Gerritsen, Amry E.; Goddard, Audrey; Godowski, Paul J.; Gurney, Austin L.; Gherwood, Steven; Smith, Victoria; Stewart, Timothy A.; Tumas, Daniel; Watanabe, Colin K.; Wood, William I.; Zhang, Zemin (Genentech, Inc., USA). PCT Int. Appl. WO 2001040466 A2 20010607, 813 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US32678 20001201. PRIORITY: WO 1999-US28301 19991201; WO 1999-US28551 19991202; WO 1999-US28565 19991202; WO 1999-US30095 19991216; WO 1999-US30999 19991220; WO 1999-US31274 19991230; WO 2000-US277 20000106; WO 2000-US3565 20000211; WO 2000-US4342 20000218; WO 2000-US4914 20000224; WO 2000-US5601 20000301; US 2000-2000/PV18720W 20000303; WO 2000-US6884 20000315; WO 2000-US7532 20000321; WO 2000-US13705 20000517.

AB The present invention is directed to novel polypeptides and to nucleic acid mols. encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. The proteins show overexpression in cancer and may of diagnostic use. Certain of the proteins were found to form complexes with one another.

L6 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

2000:881316 Document No. 134:37955 Methods and compositions for inhibiting neoplastic cell growth with utilization of chimeric polypeptides of PRO184 and PRO1186. Ashkenazi, Avi J.; Hillan, Kenneth J.; Napier, Mary A.; Watanabe, Colin K.; Wood, William I. (Genentech, Inc., USA). PCT Int. Appl. WO 2000075327 A1 20001214, 104 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US4914 20000224. PRIORITY: WO 1999-US12252 19990602; US 1999-PV145698 19990726; WO 2000-US219 20000105.

AB The present invention concerns methods and compns. for inhibiting neoplastic cell growth. In particular, the present invention concerns antitumor compns. and methods for the treatment of tumors. The invention further concerns screening methods for identifying growth inhibitory, e.g., antitumor compds. The present invention is directed to novel polypeptides and to nucleic acid mols. encoding those polypeptides which include human PRO184 and PRO1186. These proteins were effectively expressed in Escherichia coli and yeast and mammalian cells and baculovirus cells. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols.

comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. The expressed PRO184 and PRO1186 proteins may include or exclude their signal peptides or extracellular domains. These antibodies may be humanized or monoclonal. This involves the utilization of an epitope tag. In situ hybridization was employed to detect PRO1186 gene expression in ovarian cortical stroma and in leydig cells of testis. Applications for drug screening and rational drug design are described in addition to a labeled container and package insert describing the effect. Here, an antitumor assay involving antiproliferation is described. Applications for treatment of breast cancer, ovarian cancer, renal cancer, colorectal cancer, uterine cancer, prostate cancer, lung cancer, bladder cancer, CNS cancer, melanoma and leukemia are all described.

```
=> s "zven2"
L7            4 "ZVEN2"

=> s l7 and antibod?
L8            4 L7 AND ANTIBOD?

=> dup remove l8
PROCESSING COMPLETED FOR L8
L9            4 DUP REMOVE L8 (0 DUPLICATES REMOVED)

=> d l9 1-4 cbib abs

L9   ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2006 ACS on STN
2004:331918   Document No. 140:355252  Zven1 and Zven2 proteins
     associated with inflammatory bowel diseases and their use in diagnosis and
     therapy.  Thompson, Penny J.; Sheppard, Paul O. (Zymogenetics, Inc., USA).
       PCT Int. Appl. WO 2004032850 A2 20040422, 147 pp.  DESIGNATED STATES: W:
     AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR,
     CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID,
     IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD,
     MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE,
     SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW;
     RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB,
     GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR.  (English).
     CODEN: PIXXD2.  APPLICATION: WO 2003-US31562 20031007.  PRIORITY: US
     2002-416718P 20021007; US 2002-416719P 20021007; US 2002-434116P 20021216;
     US 2002-433918P 20021216; US 2003-508603P 20031003; US 2003-508614P
     20031003.
AB   Two proteins, Zven1 (prokineticin 2) and Zven2 (prokineticin 2)
     associated with inflammatory diseases of the intestines are identified as
     targets for the treatment of these diseases.  Antagonists of these
     proteins, including antibodies, can be used to induce secretion
     of chemokines to lessen inflammation.  Zven1 and Zven2 stimulate
     the release of chemokines including MIP-2, stimulate neutrophil
     chemotaxis, and angiogenesis, and slow gastric emptying.  Two G
     protein-coupled receptors for the proteins GPCR73 and GPCR73b are
     identified.

L9   ANSWER 2 OF 4  CAPLUS  COPYRIGHT 2006 ACS on STN
2004:308514   Document No. 140:332503  Protein and cDNA sequences of human
     protein Zven1 and Zven2, and use for treating intestinal
     motility disorder.  Thompson, Penny J.; Lewis, Katherine E.; Jaspers,
     Stephen R.; Garcia, Richard M.; West, Robert R.; Holderman, Susan D.;
     Chan, Chung (Zymogenetics, Inc., USA).  PCT Int. Appl. WO 2004031367 A2
     20040415, 143 pp.  DESIGNATED STATES: W:  AE, AG, AL, AM, AT, AU, AZ, BA,
     BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE,
     ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR,
     KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO,
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NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US31714 20031007. PRIORITY: US 2002-416719P 20021007; US 2002-416718P 20021007; US 2002-434116P 20021216; US 2002-433918P 20021216.

AB The present invention provides protein and cDNA sequences of human protein Zven1 and Zven2. The present invention provides methods of using Zven1 and Zven2 polypeptides to increase chemokine production The present invention also provides methods for treating intestinal motility disorders and improving gastrointestinal function with Zven1 and Zven2 polypeptides.

L9 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

2003:454937 Document No. 139:48221 Protein and cDNA sequence of human Zven proteins. Sheppard, Paul O.; Bishop, Paul D. (ZymoGenetics, Inc., USA). U.S. Pat. Appl. Publ. US 2003108994 A1 20030612, 41 pp., Division of U.S. Ser. No. 712,529. (English). CODEN: USXXCO. APPLICATION: US 2002-212355 20020802. PRIORITY: US 1999-165905P 19991116; US 2000-184875P 20000225; US 2000-197750P 20000419; US 2000-210332P 20000607; US 2000-712529 20001114.

AB The present invention provides protein and cDNA sequences of two members of a new family of human proteins, designated as "Zven". Zven1 expression was found to be restricted to B cell, T cell, monocyte, and granulocyte cell lines. Zven1 appeared to be highly expressed in the promyelocytic cell line HL60. Zven1 inhibited the proliferation of Lewis Lung cells by about 50% below controls, whereas Zven1 treatment appeared to inhibit the proliferation of normal lung cells by about 10%.

L9 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

2001:380627 Document No. 135:1260 Protein and cDNA sequences of novel human protein Zven. Sheppard, Paul O.; Bishop, Paul D.; Whitmore, Theodore E.; Thompson, Penny P. (Zymogenetics, Inc., USA). PCT Int. Appl. WO 2001036465 A2 20010525, 97 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US31278 20001114. PRIORITY: US 1999-442164 19991116; US 2000-511879 20000225; US 2000-552203 20000419; US 2000-PV210332 20000607.

AB The present invention provides protein and cDNA sequences of two members of a novel human gene family Zven, designated "Zven1" and "Zven2 ". Tissue distribution of the mRNA for the novel Zven1 fragment is restricted to B cell, T cell, monocyte and granulocyte cell lines. The Zven1 gene, which resides in human chromosome 3p21.1 - 3p14.3, is expressed in testicular tissue and peripheral blood lymphocytes. The present invention further includes agonists, antagonists, variants, antibodies and host cells expressing the cDNA encoding the novel Zven peptide.

```
=> s prokineticin 1
L10         109 PROKINETICIN 1

=> s l10 and antibod?
L11           9 L10 AND ANTIBOD?

=> dup remove l11
PROCESSING COMPLETED FOR L11
L12           9 DUP REMOVE L11 (0 DUPLICATES REMOVED)
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=> d l12 1-9 cbib abs

L12 ANSWER 1 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2006:622647 Document No.: PREV200600641766. Prokineticin polypeptides, related compositions and methods. Anonymous; Zhou, Qun-Yong [Inventor]; Ehlert, Frederick J. [Inventor]. Irvine, CA USA. ASSIGNEE: The Regents of the University of California. Patent Info.: US 07052674 20060530. Official Gazette of the United States Patent and Trademark Office Patents, (MAY 30 2006)
CODEN: OGUPE7. ISSN: 0098-1133. Language: English.
AB The invention provides isolated polypeptides that stimulate gastrointestinal smooth muscle contraction, including human prokineticin 1 and human prokineticin 2 polypeptides, and functional fragments and modifications thereof. Also provided are methods of stimulating gastrointestinal smooth muscle contraction in a mammal, by administering to the mammal an effective amount of a prokineticin polypeptide. The invention also provides nucleic acid molecules encoding a prokineticin polypeptide, and antibodies that selectively bind a prokineticin polypeptide. Further provided are methods of identifying a prokineticin receptor ligand, agonist or antagonist.

L12 ANSWER 2 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
2006423481 EMBASE Sensitization of transient receptor potential vanilloid 1 by the prokineticin receptor agonist Bv8. Vellani V.; Colucci M.; Lattanzi R.; Giannini E.; Negri L.; Melchiorri P.; McNaughton P.A.. P.A. McNaughton, Department of Pharmacology, Cambridge University, Tennis Court Road, Cambridge CB2 1PD, United Kingdom. pam42@cam.ac.uk. Journal of Neuroscience Vol. 26, No. 19, pp. 5109-5116 2006.
Refs: 35.
ISSN: 0270-6474. E-ISSN: 0270-6474. CODEN: JNRSDS
Pub. Country: United States. Language: English. Summary Language: English.
Entered STN: 20060912. Last Updated on STN: 20060912
AB Small mammalian proteins called the prokineticins [prokineticin 1 (PK1) and PK2] and two corresponding G-protein-coupled receptors [prokineticin receptor 1 (PKR1) and PKR2] have been identified recently, but the physiological role of the PK/PKR system remains mostly unexplored. Bv8, a protein extracted from frog skin, is a convenient and potent agonist for both PKR1 and PKR2, and injection of Bv8 in vivo causes a potent and long-lasting hyperalgesia. Here, we investigate the cellular basis of hyperalgesia caused by activation of PKRs. Bv8 caused increases in [Ca](i) in a population of isolated dorsal root ganglion (DRG) neurons, which we identified as nociceptors, or sensors for painful stimuli, from their responses to capsaicin, bradykinin, mustard oil, or proteases. Bv8 enhanced the inward current carried by the heat and capsaicin receptor, transient receptor potential vanilloid 1 (TRPV1) via a pathway involving activation of protein kinase Cε (PKCε), because Bv8 caused translocation of PKCε to the neuronal membrane and because PKC antagonists reduced both the enhancement of current carried by TRPV1 and behavioral hyperalgesia in rodents. The neuronal population expressing PKRs consisted partly of small peptidergic neurons and partly of neurons expressing the N52 marker for myelinated fibers. Using single-cell reverse transcriptase-PCR, we found that mRNA for PKR1 was mainly expressed in small DRG neurons. Exposure to GDNF (glial cell line-derived neurotrophic factor) induced de novo expression of functional receptors for Bv8 in a nonpeptidergic population of neurons. These results show that prokineticin receptors are expressed in nociceptors and cause heat hyperalgesia by sensitizing TRPV1 through activation of PKCε. The results suggest a role for prokineticins in physiological inflammation and hyperalgesia. Copyright .COPYRGT. 2006 Society for Neuroscience.

L12 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2006:497171 Document No.: PREV200600503491. The prokineticin receptor 1 mediates inhibition of spontaneous giant contractions in the murine

proximal colon. Hoogerwerf, Willermintje A.. Gastroenterology, (APR 2006) Vol. 130, No. 4, Suppl. 2, pp. A288.
Meeting Info.: Digestive Disease Week Meeting/107th Annual Meeting of the American-Gastroenterological-Association. Los Angeles, CA, USA. May 19 -24, 2006. Amer Gastroenterol Assoc Inst.
CODEN: GASTAB. ISSN: 0016-5085. Language: English.

AB Background: Prokineticins are novel peptides with reported effects on gastrointestinal contractility, We previously showed prokineticin receptor 1 (PKR1) expression in the myenteric plexus of the mouse proximal colon. The aims of this study were (1) to characterize the cell types expressing PKR1 (2) to determine the effect of prokineticin 1 on mouse proximal colon contractility and (3) to determine the effect of prokineticin 1 on nitric oxide release in vitro.
Methods: Immunohistochemistry was used to characterize the cell types expressing PKR1 by using double-labeling immunofluorescence in longitudinal muscle myenteric plexus (LMMP) preparations with antibodies to HuC/D (neuronal marker) and neuronal nitric oxide synthase (marker for myenteric inhibitory neurons). Proximal colon rings from adult male C57BLK6/J mice were used for circular muscle recordings in organ baths. The effect of PKC on nitric oxide release was determined from LMMP cultures using a commercial nitric oxide assay kit. Results: Quantification analysis showed that 23.8 +/- 1.85% of all HuC/D-immunoreactive neurons express PKR1 (504 neurons, 3 mice). Of PKR1-immunoreactive neurons, 7.7 +/- 1.02% express nNOS-immunoreactivity (233 neurons, 3 mice). Conversely, 5.6 +/- 1.98% of nNOS-IR neurons expressed PKR1-IR (318 neurons, 3 mice). PKC suppressed giant contractions (GC) in circular muscle. PKC 1 mM, 10 mM and 100 mM, significantly decreased the frequency from 2.22 +/- 0.48/5 at baseline to 0.89 +/- 0.20, 0.56 +/- 0.30 and 0.22 +/- 0.15 GC/5 minutes, respectively ($n=8$, $P < 0.05$). PKC 1 mM, 10 mM and 100 mM, significantly decreased the amplitude from 5.12 +/- 1.05 to 3.25 +/- 0.85, 2.20 +/- 0.80 and 1.43 +/- 0.40 mN, respectively ($n=8$, $P < 0.05$). PKC 100 mM did not significantly alter the frequency or amplitude in the setting of 1-NAME. Under non-adrenergic, non-cholinergic conditions, PKC 100 mM significantly decreased the amplitude of the contractions from 3.54 +/- 1.13 mN to 1.04 +/- 0.27 mN ($n=5$; $P < 0.05$). PKC stimulated nitric oxide release from LMMP cultures in a dose-dependent manner in vitro: PKC 1 mM, 10 mM and 100 mM produced 0.59 +/- 0.22 mMol/10 mg tissue weight, 1.19 +/- 0.28 mMol/10 mg tissue weight, and 1.55 +/- 0.39 mMol/10 mg tissue weight, respectively. This effect was blocked by 1-NAME. Conclusion: PKR1 mediates an inhibitory effect on spontaneous giant contractions in the routine colon, most likely through direct or indirect stimulation of nitric oxide release. PKR1 and its natural ligand, prokineticin 1, may be important for modulation of colonic motility.

L12 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2005:216909 Document No. 142:291401 Use of agents that reduce the effect of prokineticin 1 on a prokineticin receptor for the treatment of menorrhagia, dysmenorrhea or endometriosis. Jabbour, Henry Nicolas; Millar, Robert Peter (Ardana Bioscience Limited, UK). PCT Int. Appl. WO 2005021750 A1 20050310, 76 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-GB3600 20040824. PRIORITY: GB 2003-20238 20030829.

AB A method of combating menorrhagia, dysmenorrhea or endometriosis in a female individual is disclosed, the method comprising administering to the individual at least one agent that reduces the effect of prokineticin 1 on a prokineticin receptor.

L12 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2004:331918 Document No. 140:355252 Zven1 and Zven2 proteins associated with inflammatory bowel diseases and their use in diagnosis and therapy. Thompson, Penny J.; Sheppard, Paul O. (Zymogenetics, Inc., USA). PCT Int. Appl. WO 2004032850 A2 20040422, 147 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US31562 20031007. PRIORITY: US 2002-416718P 20021007; US 2002-416719P 20021007; US 2002-434116P 20021216; US 2002-433918P 20021216; US 2003-508603P 20031003; US 2003-508614P 20031003.
AB Two proteins, Zven1 (prokineticin 2) and Zven2 (prokineticin 2) associated with inflammatory diseases of the intestines are identified as targets for the treatment of these diseases. Antagonists of these proteins, including antibodies, can be used to induce secretion of chemokines to lessen inflammation. Zven1 and Zven2 stimulate the release of chemokines including MIP-2, stimulate neutrophil chemotaxis, and angiogenesis, and slow gastric emptying. Two G protein-coupled receptors for the proteins GPCR73 and GPCR73b are identified.

L12 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2003:796869 Document No. 139:303008 Protein and cDNA sequences of human G protein-coupled receptor (GPCR) 192 and related ligands prokineticin 1 and 2, and use thereof in screening for more novel ligands for therapeutic uses. Buckholz, Thomas; Vandenberg, Mark; Pellegrino, Carla; Heitmeier, Stefan; Taylor, Ian; Gedrich, Richard (Bayer Pharmaceuticals Corporation, USA). PCT Int. Appl. WO 2003083073 A2 20031009, 76 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US9522 20030328. PRIORITY: US 2002-368849P 20020328.
AB The present invention relates to methods for the identification and characterization of polypeptides with ligand activity for the G protein coupled receptor (GPCR) 192. Specifically, disclosed are protein and cDNA sequences of human GPCR 192 and its ligands prokineticin 1 (PROK1) and prokineticin 2 (PROK2). Also provided but not claimed are protein and cDNA sequences of mouse GPCR 73. The invention encompasses the use of the ligands in combination with the receptor for the development of assays/kits for the identification of mols. that affect the ability of the ligand to interact with the receptor. In addition, the invention relates to mols. affecting the expression of the ligands (e.g., antisense DNA, ribozymes, antibodies) that may modulate the activity of the receptor. The invention also encompasses the use of the ligands and their derivs., mols. affecting ligand expression, and compds. that modulate receptor activity for the treatment of disorders involving GPCR 192, such as those of the central nervous system, metabolic disorders (e.g., pancreatic disorders), gastrointestinal disorders, immune disorders, and cancer.

L12 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2003:633928 Document No. 139:175555 Drug screening for inhibitors of peptide ligands for G-protein-coupled receptors ZAQ and 15E as angiogenesis inhibitors. Ohtaki, Tetsuya; Masuda, Yasushi; Takatsu, Yoshihiro (Takeda Chemical Industries, Ltd., Japan). PCT Int. Appl. WO 2003066860 A1 20030814, 308 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE,

ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (Japanese). CODEN: PIXXD2. APPLICATION: WO 2003-JP1057 20030203. PRIORITY: JP 2002-27299 20020204.

AB Provided are a method and kit for screening compds. inhibiting the activity of novel peptide ligands for two orphan G-protein-coupled receptors ZAQ and 15E. Such compds., antisense nucleic acids or antibodies, are usable as, for example, angiogenesis inhibitors in diagnosis, prevention, and therapy for cancer, polycystic ovary syndrome, ovary overstimulation, etc. The amino acid sequences of those peptides, human, mouse, rat, and bovine ZAQ ligand peptide, snake venom MITI and human and other mammalian homolog (Bv8 peptide), are provided. Endocrine gland-derived vascular endothelial growth factor (EG-VEGF, identical to prokineticin 1) is a novel peptide recently identified as a selective mitogen for endocrine gland endothelial cells. The present study demonstrates that EG-VEGF/prokineticin 1 and a peptide closely related to EG-VEGF, prokineticin 2, are cognate ligands of two orphan G-protein-coupled receptors designated ZAQ (= EG-VEGF/PK-R1) and 15E (= EG-VEGF/PK-R2). EG-VEGF/prokineticin 1 and prokineticin 2 induced a transient increase in intracellular calcium ion concentration ([Ca2+]i) with nanomolar potency in Chinese hamster ovary (CHO) cells expressing EG-VEGF/PK-R1 and -R2 and bind to these cells with high affinity and with different receptor selectivity. EG-VEGF/prokineticins provoke rapid phosphorylation of p44/42 MAP kinase and DNA synthesis in the bovine adrenal capillary endothelial cells (BACE). The mRNAs of both EG-VEGF/PK-R1 and -R2 were expressed in BACE. The identification of the receptors for EG-VEGF/prokineticins may provide a novel mol. basis for the regulation of angiogenesis in endocrine glands.

L12 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2003:377016 Document No. 138:380374 Methods and materials relating to prokineticin-like polypeptides and polynucleotides. Ghosh, Malabika J.; Tang, Tom Y.; Liu, Chenghua; Drmanac, Radoje T. (Hyseq, Inc., USA; Nuvelo Inc.). PCT Int. Appl. WO 2003040326 A2 20030515, 132 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-US35465 20021104. PRIORITY: US 2001-343902P 20011102.

AB The invention provides novel polynucleotides and polypeptides encoded by such polynucleotides and mutants or variants thereof that correspond to novel human prokineticin-like polypeptide. Polynucleotide of the invention comprises of nucleic acid sequence isolated from cDNA library prepared from human bone marrow (Clontech) mRNA for prokineticin-like polypeptide. Prokineticin-like protein is homologous to human, mouse and toad Bv8 proteins, human prokineticin 1 and 2, and Mamba intestinal toxin (MIT). Other aspects of the invention include vectors containing processes for producing novel human prokineticin polypeptide, and antibodies specific for such a polypeptide. The present invention claimed cDNA and protein sequences for the prokineticin-like protein, but the cDNA sequence was not made available on publication of the patent application.

L12 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2002:353486 Document No. 136:364202 Identification, preparation, characterization, and therapeutic use of prokineticin polypeptides that stimulate the gastrointestinal tract. Zhou, Qun-Yong; Ehlert, Frederick J. (The Regents of the University of California, USA). PCT Int. Appl. WO

2002036625 A2 20020510, 86 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US47969 20011101. PRIORITY: US 2000-245882P 20001103.

AB The invention provides isolated polypeptides that stimulate gastrointestinal smooth muscle contraction, including human prokineticin 1 and human prokineticin 2 polypeptides, and functional fragments and modifications thereof. Also provided are methods of stimulating gastrointestinal smooth muscle contraction in a mammal, by administering to the mammal an effective amount of a prokineticin polypeptide. The invention also provides nucleic acid mols. encoding a prokineticin polypeptide, and antibodies that selectively bind a prokineticin polypeptide. Further provided are methods of identifying a prokineticin receptor ligand, agonist or antagonist.

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L13 0 "HUMAN TANGO 266"

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L15 2 DUP REMOVE L14 (0 DUPLICATES REMOVED)

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L15 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
2003:98030 Document No. 138:148750 Genes encoding human rat, and mouse proteins having prognostic, diagnostic, preventive, therapeutic, and other uses. Holtzman, Douglas A.; Barnes, Thomas M. (USA). U.S. Pat. Appl. Publ. US 2003027998 A1 20030206, 745 pp., Cont.-in-part of U. S. Ser. No. 667,751. (English). CODEN: USXXCO. APPLICATION: US 2001-796753 20010301. PRIORITY: US 1998-183175 19981030; US 1998-223546 19981230; US 1998-224246 19981230; US 1998-223094 19981230; US 1999-259388 19990226; US 1999-336536 19990618; US 1999-365164 19990730; US 1999-399723 19990920; US 1999-409634 19990930; US 1999-471179 19991223; US 1999-474072 19991229; US 1999-474071 19991229; US 2000-514010 20000225; US 2000-516745 20000301; US 2000-597993 20000619; US 2000-599596 20000622; US 2000-630334 20000731; US 2000-665666 20000920; US 2000-667751 20000921.

AB The invention provides 51 nucleic acid mols. and their encoded polypeptide mols. isolated from human, mouse, and rat tissues. Protein motifs and domains, hydropathy plots, chromosomal mapping, and tissue expression profiles are provided for each of the proteins. TANGO 266 is shown to modulate the proliferation and differentiation of macrophages and hematopoietic progenitor cells. The invention also provides antisense nucleic acid mols., expression vectors containing the nucleic acid mols. of the invention, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a nucleic acid mol. of the invention has been introduced or disrupted. The invention still further provides isolated polypeptides, fusion polypeptides, antigenic peptides and antibodies. Diagnostic, screening and therapeutic methods utilizing compns. of the invention are also provided.

L15 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
2000:628148 Document No. 133:218529 Human secreted proteins and nucleic acids encoding them. Barnes, Thomas M.; Holtzman, Douglas A.; Sharp, John D.; Fraser, Christopher C. (Millennium Pharmaceuticals, Inc., USA). PCT

Int. Appl. WO 2000052022 A1 20000908, 176 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US5226 20000301. PRIORITY: US 1999-PV122458 19990301.

AB The invention provides isolated cDNA mols., designated TANGO 216, TANGO 261, TANGO 262, TANGO 266, and TANGO 267 from mouse and human tissues. These nucleic acid mols. encode wholly secreted and transmembrane proteins. Tissue expression and gene mapping data are also provided. The invention also provides antisense nucleic acid mols., expression vectors containing the nucleic acid mols. of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a nucleic acid mol. of the invention has been introduced or disrupted. The invention still further provides isolated polypeptides, fusion polypeptides, antigenic peptides and antibodies. Diagnostic, screening and therapeutic methods utilizing compns. of the invention are also provided.

=> s (ferrara n?/au or watanabe c?/au or wood w?/au or shek t?/au)
L16 11862 (FERRARA N?/AU OR WATANABE C?/AU OR WOOD W?/AU OR SHEK T?/AU)

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L18 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
2005:534473 Document No. 143:472697 Vascular bed-specific signaling and angiogenesis. Ferrara, Napoleone; Lin, Rui; LeCouter, Jennifer (Department of Molecular Oncology, Genentech Inc., South San Francisco, CA, USA). Endothelial Cells in Health and Disease, 203-216. Editor(s): Aird, William C. Taylor & Francis: Boca Raton, Fla. ISBN: 0-8247-5424-7 (English) 2005. CODEN: 69GXXN.

AB A review focuses on the potential role of EG-VGF (vascular growth factor) in the cyclic ovarian angiogenesis, and possibly in the pathophysiol. of other steroidogenic organs, such as adrenal and placenta.

L18 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
2004:780858 Document No. 141:273369 Compositions comprising human and mouse Bv8 and EG-VEGF with hematopoietic and immune activity and therapeutic uses for blood diseases and immune disorders. Ferrara, Napoleone; Lecouter, Jennifer (Genentech, Inc., USA). PCT Int. Appl. WO 2004081229 A2 20040923, 161 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-US7622 20040312. PRIORITY: US 2003-454462P 20030312; US 2003-511390P 20031014.

AB The present invention relates to the novel expression and activities of Bv8 and EG-VEGF in hematopoietic stem cells (HSCs), lineage-committed blood progenitor cells, and lymphocytes. In particular,

Bv8, EG-VEGF, and their receptors are expressed in bone marrow HSCs, peripheral blood leukocytes (PBLs), as well as many hematol. malignant cell lines. Bv8 and EG-VEGF are capable of promoting colony formation of bone marrow mononuclear cells and spleen-derived progenitor cells, increasing populations of white blood cells, and promoting activation of B lymphocytes and T lymphocytes. BvS8 nucleic acids and polypeptides, EG-VEGF nucleic acids and polypeptides, or combinations thereof can be used in a number of assays and in diagnosis and treatment of conditions associated with hematopoiesis, neutropenias, immunodeficiency disorders, and autoimmune disorders.

L18 ANSWER 3 OF 18 MEDLINE on STN DUPLICATE 1

2004599383. PubMed ID: 15548611. Bv8 and endocrine gland-derived vascular endothelial growth factor stimulate hematopoiesis and hematopoietic cell mobilization. LeCouter Jennifer; Zlot Constance; Tejada Max; Peale Franklin; Ferrara Napoleone. (Department of Physiology, Genentech, Inc., South San Francisco, CA 94080, USA.. lecouter@gene.com) . Proceedings of the National Academy of Sciences of the United States of America, (2004 Nov 30) Vol. 101, No. 48, pp. 16813-8. Electronic Publication: 2004-11-17. Journal code: 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB Bv8 and endocrine-gland-derived VEGF (EG-VEGF), or prokineticins, are two highly related, secreted proteins that we previously described as selective angiogenic mitogens. Here we describe the expression and functional characterization of Bv8 in peripheral blood cells, notably monocytes, neutrophils, and dendritic cells, and in the bone marrow. In human and mouse, the two Bv8 G protein-coupled receptors are expressed in hematopoietic stem cells and specific mature blood cells, including lymphocytes. Bv8 is highly expressed by neutrophils at sites of inflammation and can stimulate migration of monocytes, in a pertussis toxin-sensitive manner. Bv8, or EG-VEGF that shares the same receptors, increased numbers of colony-forming units granulocytic and monocytic in cultures of human or mouse hematopoietic stem cells. Systemic in vivo exposure to Bv8 or EG-VEGF resulted in significant increases in total leukocyte, neutrophil, and monocyte counts. Additionally, adenovirus (Av)Bv8 or AvEG-VEGF delivered just before 5-fluorouracil injury promoted the survival of hematopoietic cells and enhanced progenitor mobilization. In conclusion, Bv8 can promote survival and differentiation of the granulocytic and monocytic lineages. Bv8 potentially modulates growth, survival, and function of cells of the innate and adaptive immune systems, possibly through autocrine or paracrine signaling mechanisms.

L18 ANSWER 4 OF 18 MEDLINE on STN DUPLICATE 2

2004388609. PubMed ID: 15292351. Human endocrine gland-derived vascular endothelial growth factor: expression early in development and in Leydig cell tumors suggests roles in normal and pathological testis angiogenesis. Samson Michel; Peale Franklin V Jr; Frantz Gretchen; Rioux-Leclercq Nathalie; Rajpert-De Meyts Ewa; Ferrara Napoleone. (Department of Molecular Oncology, Genentech, Inc., South San Francisco, California 94080, USA.) The Journal of clinical endocrinology and metabolism, (2004 Aug) Vol. 89, No. 8, pp. 4078-88. Journal code: 0375362. ISSN: 0021-972X. Pub. country: United States. Language: English.

AB Angiogenesis is essential for tumor growth and metastasis. A new human angiogenic mitogen, endocrine gland-derived vascular endothelial growth factor (EG-VEGF), has been recently identified; its expression pattern is restricted to endocrine glands, with the highest expression in testis. We used in situ hybridization and newly generated monoclonal antibodies to investigate the expression of EG-VEGF in normal human prenatal and adult testis and in 48 human testicular tumors of different subtypes. We found that EG-VEGF was expressed from 14 wk until birth in human fetal testis. In the adult testis, EG-VEGF was strongly expressed only in Leydig cells. In testicular tumors, EG-VEGF was expressed specifically in Leydig cell tumors, whereas germ

cell-derived neoplasms, including carcinoma in situ, seminoma, and nonseminomatous germ cell tumors, were negative for this antigen. In contrast, VEGF, another powerful angiogenic factor, was expressed in seminoma, but very weakly in Leydig cell tumors. Interestingly, we found that Leydig cell tumors presented vessel surface density 3.2-fold higher than seminoma. These findings argue that human EG-VEGF may play a role in angiogenesis both during the early endocrine development of testis and in the adult testis as well as in Leydig cell tumor growth.

L18 ANSWER 5 OF 18 MEDLINE on STN DUPLICATE 3
2004095079. PubMed ID: 14984768. EG-VEGF and Bv8: a novel family of tissue-restricted angiogenic factors. Ferrara Napoleone; LeCouter Jennifer; Lin Rui; Peale Franklin. (Department of Molecular Oncology, Genentech Inc, South San Francisco, CA 94080, USA.. nf@gene.com) . Biochimica et biophysica acta, (2004 Mar 4) Vol. 1654, No. 1, pp. 69-78. Ref: 82. Journal code: 0217513. ISSN: 0006-3002. Pub. country: Netherlands. Language: English.

AB A novel family of angiogenic mitogens have been recently characterized. Endocrine gland-derived vascular endothelial growth factor (EG-VEGF), and the mammalian homologue of Bombina variegata peptide 8 (Bv8), are two highly related endothelial cell mitogens and chemotactic factors with restricted expression profiles and selective endothelial cell activity. These peptides share two cognate G-protein coupled receptors. The expression of human EG-VEGF occurs predominantly in steroidogenic glands. Consistent with such an expression pattern, the human EG-VEGF gene promoter has a potential binding site for steroidogenic factor (SF)-1, a pivotal element for steroidogenic-specific transcription. In the human ovary, the expression of EG-VEGF is temporally and spatially complementary to the expression of VEGF-A, both in the follicular and in the luteal phase, suggesting complementary and coordinated roles of these molecules in ovarian angiogenesis. Also, EG-VEGF expression correlates with vascularity in the polycystic ovary syndrome, a leading cause of infertility. Bv8 expression is mainly restricted to the testis. The identification of these tissue-selective angiogenic factors raises the possibility that other secreted molecules with selectivity for the endothelium of other organs exist.

L18 ANSWER 6 OF 18 MEDLINE on STN DUPLICATE 4
2004255671. PubMed ID: 15153419. EG-VEGF: a novel mediator of endocrine-specific angiogenesis, endothelial phenotype, and function. Lecouter Jennifer; Lin Rui; Ferrara Napoleone. (Department of Molecular Oncology, Genentech Inc., South San Francisco, California 94080, USA.) Annals of the New York Academy of Sciences, (2004 Apr) Vol. 1014, pp. 50-7. Ref: 69. Journal code: 7506858. ISSN: 0077-8923. Pub. country: United States. Language: English.

AB Angiogenesis is the focus of therapeutic efforts to promote new vessel development in damaged tissues. Conversely, inhibiting endothelial cell growth and survival is a strategy to treat various proliferative diseases. Much evidence indicates that VEGF is a key mediator of angiogenesis. Recently, a novel angiogenic mitogen with tissue-specific expression and target selectivity was characterized. Human endocrine gland derived vascular endothelial growth factor (EG-VEGF) is selectively expressed in steroidogenic glands and promotes growth of endocrine gland endothelium. The identification of tissue-selective angiogenic factors raises the possibility that other secreted molecules in this class exist. The potential advantage of tissue-specific angiogenic therapeutics may be the reduction of systemic side effects. Additionally, these peptides or their receptors may be attractive targets for inhibition in several disorders.

L18 ANSWER 7 OF 18 MEDLINE on STN DUPLICATE 5
2003106208. PubMed ID: 12604792. The endocrine-gland-derived VEGF homologue Bv8 promotes angiogenesis in the testis: Localization of Bv8

receptors to endothelial cells. LeCouter Jennifer; Lin Rui; Tejada Max; Frantz Gretchen; Peale Franklin; Hillan Kenneth J; Ferrara Napoleone. (Department of Molecular Oncology, Genentech, Inc., South San Francisco, CA 94080, USA.) Proceedings of the National Academy of Sciences of the United States of America, (2003 Mar 4) Vol. 100, No. 5, pp. 2685-90. Electronic Publication: 2003-02-25. Journal code: 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB We recently identified an angiogenic mitogen, endocrine-gland-derived vascular endothelial growth factor (EG-VEGF), with selective activity for endothelial cells of endocrine tissues. Here we describe the characterization of a highly related molecule, Bv8, also known as prokineticin-2. Human Bv8 shares 60% identity and 75% similarity with EG-VEGF. The human and mouse Bv8 genes share a common structure. Like EG-VEGF, Bv8 is able to induce proliferation, survival and migration of adrenal cortical capillary endothelial cells. Bv8 gene expression is induced by hypoxic stress. Bv8 expression occurs predominantly in the testis and is largely restricted to primary spermatocytes. Adenoviral delivery of Bv8 or EG-VEGF to the mouse testis resulted in a potent angiogenic response. We have localized the expression of the Bv8EG-VEGF receptors within the testis to vascular endothelial cells. The testis exhibits relatively high turnover of endothelial cells. Therefore, Bv8 and EG-VEGF, along with other factors such as VEGF-A, may maintain the integrity and also regulate proliferation of the blood vessels in the testis.

L18 ANSWER 8 OF 18 MEDLINE on STN DUPLICATE 6

2003223990. PubMed ID: 12746324. Mouse endocrine gland-derived vascular endothelial growth factor: a distinct expression pattern from its human ortholog suggests different roles as a regulator of organ-specific angiogenesis. LeCouter Jennifer; Lin Rui; Frantz Gretchen; Zhang Zemin; Hillan Kenneth; Ferrara Napoleone. (Department of Molecular Oncology, Genentech Inc., South San Francisco, California 94080, USA.) Endocrinology, (2003 Jun) Vol. 144, No. 6, pp. 2606-16. Journal code: 0375040. ISSN: 0013-7227. Pub. country: United States. Language: English.

AB We recently described human endocrine gland-derived vascular endothelial growth factor (EG-VEGF) as an endothelial cell mitogen with a novel selective activity and an expression pattern essentially limited to steroidogenic glands. Herein we present the identification and characterization of the mouse ortholog. The mouse cDNA and predicted amino acid sequences are, respectively, 86% and 88% identical with the human. Surprisingly, the mouse EG-VEGF transcript is predominantly expressed in liver and kidney. A comparison of human and mouse EG-VEGF promoter sequences revealed a potential binding site for NR5A1, which is known to be a pivotal element for steroidogenic-specific transcription, in the human but not mouse promoter. In situ hybridization studies localized expression of mouse EG-VEGF mRNA to hepatocytes and renal tubule cells. Interestingly, capillary endothelial cells in these sites share several common structural features with those found in steroidogenic glands. Within liver and kidney, EG-VEGF receptor expression was largely restricted to endothelial cells. Mouse EG-VEGF promoted proliferation and survival of endothelial cells. We propose that mouse EG-VEGF, like human EG-VEGF, plays a role in regulating the phenotype and growth properties of endothelial cells within distinct capillary beds.

L18 ANSWER 9 OF 18 MEDLINE on STN DUPLICATE 7

2003235306. PubMed ID: 12759245. Differential expression of the angiogenic factor genes vascular endothelial growth factor (VEGF) and endocrine gland-derived VEGF in normal and polycystic human ovaries. Ferrara Napoleone; Frantz Gretchen; LeCouter Jennifer; Dillard-Telm Lisa; Pham Thinh; Draksharapu Aparna; Giordano Thomas; Peale Franklin. (Department of Molecular Oncology, Genentech Incorporated, South San Francisco, California 94080, USA.) The American journal of pathology,

(2003 Jun) Vol. 162, No. 6, pp. 1881-93. Journal code: 0370502. ISSN: 0002-9440. Pub. country: United States. Language: English.

AB Angiogenesis is a key aspect of the dynamic changes occurring during the normal ovarian cycle. Hyperplasia and hypervascularity of the ovarian theca interna and stroma are also prominent features of the polycystic ovary syndrome (PCOS), a leading cause of infertility. Compelling evidence indicated that vascular endothelial growth factor (VEGF) is a key mediator of the cyclical corpus luteum angiogenesis. However, the nature of the factor(s) that mediate angiogenesis in PCOS is less clearly understood. Endocrine gland-derived (EG)-VEGF has been recently identified as an endothelial cell mitogen with selectivity for the endothelium of steroidogenic glands and is expressed in normal human ovaries. In the present study, we compared the expression of EG-VEGF and VEGF mRNA in a series of 13 human PCOS and 13 normal ovary specimens by in situ hybridization. EG-VEGF expression in normal ovaries is dynamic and generally complementary to VEGF expression in both follicles and corpora lutea. A particularly high expression of EG-VEGF was detected in the Leydig-like hilus cells found in the highly vascularized ovarian hilus. In PCOS ovaries, we found strong expression of EG-VEGF mRNA in theca interna and stroma in most of the specimens examined, thus spatially related to the new blood vessels. In contrast, VEGF mRNA expression was most consistently associated with the granulosa cell layer and sometimes the theca, but rarely with the stroma. These findings indicate that both EG-VEGF and VEGF are expressed in PCOS ovaries, but in different cell types at different stages of differentiation, thus suggesting complementary functions for the two factors in angiogenesis and possibly cyst formation.

L18 ANSWER 10 OF 18 MEDLINE on STN DUPLICATE 8

2003461872. PubMed ID: 14522467. EG-VEGF and Bv8. a novel family of tissue-selective mediators of angiogenesis, endothelial phenotype, and function. LeCouter Jennifer; Ferrara Napoleone. (Department of Molecular Oncology, Genentech Inc., 1 DNA Way, South San Francisco, CA 94080, USA.) Trends in cardiovascular medicine, (2003 Oct) Vol. 13, No. 7, pp. 276-82. Ref: 80. Journal code: 9108337. ISSN: 1050-1738. Pub. country: United States. Language: English.

AB Angiogenic molecules are the focus of therapeutic efforts to promote new vessel development in ischemic or damaged tissue and, conversely, to inhibit endothelial cell growth and survival in proliferative disease. Two novel angiogenic mitogens have been characterized recently. Endocrine gland-derived vascular endothelial growth factor (EG-VEGF) and the mammalian homologue of Bombina variegata peptide 8 (Bv8) are endothelial cell mitogens and chemotactic factors with restricted expression profiles and selective endothelial cell activity. These highly related peptides share two cognate G-protein-coupled receptors that are homologous to the neuropeptide Y receptor. The identification of tissue-selective angiogenic factors raises the possibility that other secreted molecules in this class exist. The potential advantage of tissue-specific angiogenic therapeutics may be the reduction of systemic side effects. Additionally, these peptides or their receptors may be attractive targets for inhibition in several disorders.

L18 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN

2002:10528 Document No. 136:65270 Protein and cDNA sequences encoding human EG-VEGF protein and methods of use. Ferrara, Napoleone; Watanabe, Colin; Wood, William I. (Genentech, Inc., USA). PCT Int. Appl. WO 2002000711 A2 20020103, 133 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML,

MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US20116 20010622. PRIORITY: US 2000-213637P 20000623; US 2000-230978P 20000907; WO 2000-US32678 20001201.

AB The present invention is directed to novel polypeptides designated herein as EG-VEGF of human and to nucleic acid mols. encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Also provided herein are methods of screening for modulators of EG-VEGF. Furthermore, methods and related methods of treatment are described herein which pertain to regulating cellular proliferation and chemotaxis.

L18 ANSWER 12 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN

2002:964996 Document No. 138:33697 Endocrine gland-derived vascular endothelial growth factor nucleic acids and polypeptides and their biological activities and use in drug screening and therapies. Ferrara, Napoleone; Watanabe, Colin; Wood, William I.; Shek, Theresa (USA). U.S. Pat. Appl. Publ. US 2002192634 A1 20021219, 105 pp., Cont.-in-part of U.S. Ser. No. 886,242. (English). CODEN: USXXCO. APPLICATION: US 2001-27603 20011219. PRIORITY: US 1998-96146P 19980811; WO 1999-US12252 19990602; US 1999-145698P 19990726; US 1999-380137 19990825; WO 2000-US219 20000105; WO 2000-US4914 20000224; WO 2000-US8439 20000330; US 2000-213637P 20000623; US 2000-230978P 20000907; US 2000-709238 20001108; WO 2000-US32678 20001201; US 2001-886242 20010620.

AB The present invention is based on the identification and characterization of a novel, tissue-restricted, growth and differentiation factor that acts selectively on one endothelial cell type. This factor, referred to as endocrine gland-derived vascular endothelial growth factor (EG-VEGF), induces proliferation, migration, and fenestrations in capillary endothelial cells derived from endocrine glands, but has no effect on a variety of other endothelial and non-endothelial cell types tested. EG-VEGF also induces phosphorylation of kinases involved in cell proliferation or survival, including ERK1, ERK2, Akt, and eNOS. EG-VEGF nucleic acids and polypeptides can be used in a number of assays and in diagnosis and treatment of conditions associated with hormone-producing tissue. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Also provided herein are methods of screening for modulators of EG-VEGF. Furthermore, methods and related methods of treatment are described herein which pertain to regulating cellular proliferation and chemotaxis.

L18 ANSWER 13 OF 18 MEDLINE on STN DUPLICATE 9

2002139189. PubMed ID: 11751915. Characterization of endocrine gland-derived vascular endothelial growth factor signaling in adrenal cortex capillary endothelial cells. Lin Rui; LeCouter Jennifer; Kowalski Joe; Ferrara Napoleone. (Department of Molecular Oncology, Genentech Inc., South San Francisco, California 94080, USA.) The Journal of biological chemistry, (2002 Mar 8) Vol. 277, No. 10, pp. 8724-9. Electronic Publication: 2001-12-20. Journal code: 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.

AB Endocrine gland-derived vascular endothelial growth factor (EG-VEGF) has been recently identified as a mitogen specific for the endothelium of steroidogenic glands. Here we report a characterization of the signal transduction of EG-VEGF in a responsive cell type, bovine adrenal cortex-derived endothelial (ACE) cells. EG-VEGF led to a time- and dose-dependent phosphorylation of p44/42 MAPK. This effect was blocked by pretreatment

with pertussis toxin, suggesting that G alpha(i) plays an important role in mediating EG-VEGF-induced activation of MAPK signaling. The inhibitor of p44/42 MAPK phosphorylation PD 98059 resulted in suppression of both proliferation and migration in response to EG-VEGF. EG-VEGF also increased the phosphorylation of Akt in a phosphatidylinositol 3-kinase-dependent manner. Consistent with such an effect, EG-VEGF was a potent survival factor for ACE cells. We also identified endothelial nitric-oxide synthase as one of the downstream targets of Akt activation. Phosphorylation of endothelial nitric-oxide synthase in ACE cells was stimulated by EG-VEGF with a time course correlated to the Akt phosphorylation. Our data demonstrate that EG-VEGF, possibly through binding to a G-protein coupled receptor, results in the activation of MAPK p44/42 and phosphatidylinositol 3-kinase signaling pathways, leading to proliferation, migration, and survival of responsive endothelial cells.

L18 ANSWER 14 OF 18 MEDLINE on STN DUPLICATE 10
2003023758. PubMed ID: 12530695. Endocrine gland vascular endothelial growth factor (EG-VEGF) and the hypothesis of tissue-specific regulation of angiogenesis. Ferrara Napoleone; LeCouter Jennifer; Lin Rui. (Department of Molecular Oncology, Genentech Inc., South San Francisco, CA 94080, USA.. nf@gene.com) . Endocrine research, (2002 Nov) Vol. 28, No. 4, pp. 763-4. Journal code: 8408548. ISSN: 0743-5800. Pub. country: United States. Language: English.

L18 ANSWER 15 OF 18 MEDLINE on STN DUPLICATE 11
2003329369. PubMed ID: 12858543. The role of EG-VEGF in the regulation of angiogenesis in endocrine glands. LeCouter J; Lin R; Ferrara N. (Department of Molecular Oncology, Genentech, Inc., South San Francisco, California 94080, USA.) Cold Spring Harbor symposia on quantitative biology, (2002) Vol. 67, pp. 217-21. Ref: 51. Journal code: 1256107. ISSN: 0091-7451. Pub. country: United States. Language: English.

L18 ANSWER 16 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 12
2002:446084 Document No.: PREV200200446084. EG-VEGF and the hypothesis of tissue-specific regulation of angiogenesis. Ferrara, Napoleone [Reprint author]; LeCouter, Jennifer [Reprint author]; Lin, Rui [Reprint author]. Dept Molecular Oncology, Genentech Inc, South San Francisco, CA, USA. Biology of Reproduction, (2002) Vol. 66, No. Supplement 1, pp. 82. print.
Meeting Info.: 35th Annual Meeting of the Society for the Study of Reproduction. Baltimore, Maryland, USA. July 28-31, 2002.
CODEN: BIREBV. ISSN: 0006-3363. Language: English.

L18 ANSWER 17 OF 18 MEDLINE on STN DUPLICATE 13
2002233229. PubMed ID: 11969366. EG-VEGF and the concept of tissue-specific angiogenic growth factors. LeCouter Jennifer; Ferrara Napoleone. (Department of Molecular Oncology, Genentech, 1 DNA Way, South San Francisco, CA 94080, USA.) Seminars in cell & developmental biology, (2002 Feb) Vol. 13, No. 1, pp. 3-8. Ref: 68. Journal code: 9607332. ISSN: 1084-9521. Pub. country: England: United Kingdom. Language: English.
AB The endothelium of the vascular beds is extremely diverse and exquisitely distinct with respect to the specific tissue compartment served by the vessels. The molecular identity and function of the instructive signals that tailor the tissue-specific endothelial phenotype have been largely undefined. Presumably, a complex, integrated network of signals derived from the tissue parenchyma and/or stromal compartments is responsible. Recently, we identified a novel angiogenic mitogen, endocrine-gland-derived vascular endothelial growth factor, EG-VEGF, with a selective activity and very distinct expression pattern. Human EG-VEGF is expressed by steroid producing cells in the

adrenal gland, placenta, testis and ovary, and is a mitogen for endothelial cells derived from these microvascular beds. EG-VEGF may represent the first of a novel class of tissue-specific angiogenic factors that function to regulate and fine-tune endothelial cell growth, structural and functional properties. The identification of other selective angiogenic molecules will allow insight into exciting, basic developmental issues and increase our armamentarium of factors for therapeutic angiogenic and anti-angiogenic strategies.

L18 ANSWER 18 OF 18 MEDLINE on STN DUPLICATE 14
2001486108. PubMed ID: 11528470. Identification of an angiogenic mitogen selective for endocrine gland endothelium. LeCouter J; Kowalski J; Foster J; Hass P; Zhang Z; Dillard-Telm L; Frantz G; Rangell L; DeGuzman L; Keller G A; Peale F; Gurney A; Hillan K J; Ferrara N. (Department of Molecular Oncology, Genentech Inc., South San Francisco, CA 94080, USA.) Nature, (2001 Aug 30) Vol. 412, No. 6850, pp. 877-84. Journal code: 0410462. ISSN: 0028-0836. Pub. country: England: United Kingdom. Language: English.
AB The known endothelial mitogens stimulate growth of vascular endothelial cells without regard to their tissue of origin. Here we report a growth factor that is expressed largely in one type of tissue and acts selectively on one type of endothelium. This molecule, called endocrine-gland-derived vascular endothelial growth factor (EG-VEGF), induced proliferation, migration and fenestration (the formation of membrane discontinuities) in capillary endothelial cells derived from endocrine glands. However, EG-VEGF had little or no effect on a variety of other endothelial and non-endothelial cell types tested. Similar to VEGF, EG-VEGF possesses a HIF-1 binding site, and its expression is induced by hypoxia. Both EG-VEGF and VEGF resulted in extensive angiogenesis and cyst formation when delivered in the ovary. However, unlike VEGF, EG-VEGF failed to promote angiogenesis in the cornea or skeletal muscle. Expression of human EG-VEGF messenger RNA is restricted to the steroidogenic glands, ovary, testis, adrenal and placenta and is often complementary to the expression of VEGF, suggesting that these molecules function in a coordinated manner. EG-VEGF is an example of a class of highly specific mitogens that act to regulate proliferation and differentiation of the vascular endothelium in a tissue-specific manner.

=> s l16 and "PRO 1186"
L19 0 L16 AND "PRO 1186"

=> s l16 and "PRO1186"
L20 3 L16 AND "PRO1186"

=> d l20 1-3 cbib abs

L20 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
2002:773696 Document No. 137:289998 Cloning, protein and cDNA sequence of human protein PRO1186. Baker, Kevin; Chen, Jian; Goddard, Audrey; Gurney, Austin L.; Smith, Victoria; Watanabe, Colin K.; Wood, William I.; Yuan, Jean (Genentech, Inc., USA). Eur. Pat. Appl. EP 1247863 A1 20021009, 49 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL. (English). CODEN: EPXXDW. APPLICATION: EP 2002-12968 19990602. PRIORITY: US 1998-96146P 19980811; EP 1999-955293 19990602.
AB The invention relates to protein and cDNA sequence of human protein PRO1186. The invention relates to methods and vector for recombinant expression of protein PRO1186 in mammalian cell, yeast, Escherichia coli and insect cell. The invention relates to antibody against protein PRO1186 and drug screening.

L20 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
2001:417147 Document No. 135:29838 Secreted and transmembrane proteins identified by sequence comparison and cDNAs encoding them and their uses. Baker, Kevin; Beresini, Maureen; Deforge, Laura; Desnoyers, Luc; Filvaroff, Ellen; Gao, Wei Qiang; Gerritsen, Amry E.; Goddard, Audrey; Godowski, Paul J.; Gurney, Austin L.; Gherwood, Steven; Smith, Victoria; Stewart, Timothy A.; Tumas, Daniel; Watanabe, Colin K.; Wood, William I.; Zhang, Zemin (Genentech, Inc., USA). PCT Int. Appl. WO 2001040466 A2 20010607, 813 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US32678 20001201. PRIORITY: WO 1999-US28301 19991201; WO 1999-US28551 19991202; WO 1999-US28565 19991202; WO 1999-US30095 19991216; WO 1999-US30999 19991220; WO 1999-US31274 19991230; WO 2000-US277 20000106; WO 2000-US3565 20000211; WO 2000-US4342 20000218; WO 2000-US4914 20000224; WO 2000-US5601 20000301; US 2000-2000/PV18720W 20000303; WO 2000-US6884 20000315; WO 2000-US7532 20000321; WO 2000-US13705 20000517.

AB The present invention is directed to novel polypeptides and to nucleic acid mols. encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. The proteins show overexpression in cancer and may of diagnostic use. Certain of the proteins were found to form complexes with one another.

L20 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
2000:881316 Document No. 134:37955 Methods and compositions for inhibiting neoplastic cell growth with utilization of chimeric polypeptides of PRO184 and PRO1186. Ashkenazi, Avi J.; Hillan, Kenneth J.; Napier, Mary A.; Watanabe, Colin K.; Wood, William I. (Genentech, Inc., USA). PCT Int. Appl. WO 2000075327 A1 20001214, 104 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US4914 20000224. PRIORITY: WO 1999-US12252 19990602; US 1999-PV145698 19990726; WO 2000-US219 20000105.

AB The present invention concerns methods and compns. for inhibiting neoplastic cell growth. In particular, the present invention concerns antitumor compns. and methods for the treatment of tumors. The invention further concerns screening methods for identifying growth inhibitory, e.g., antitumor compds. The present invention is directed to novel polypeptides and to nucleic acid mols. encoding those polypeptides which include human PRO184 and PRO1186. These proteins were effectively expressed in Escherichia coli and yeast and mammalian cells and baculovirus cells. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. The expressed PRO184 and PRO1186 proteins may include or exclude their signal peptides or extracellular domains. These antibodies may be humanized or monoclonal. This involves the

utilization of an epitope tag. In situ hybridization was employed to detect PRO1186 gene expression in ovarian cortical stroma and in leydig cells of testis. Applications for drug screening and rational drug design are described in addition to a labeled container and package insert describing the effect. Here, an antitumor assay involving antiproliferation is described. Applications for treatment of breast cancer, ovarian cancer, renal cancer, colorectal cancer, uterine cancer, prostate cancer, lung cancer, bladder cancer, CNS cancer, melanoma and leukemia are all described.

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L21 2890679 ANTIBOD?

=> s l21 and venom black mamba snake dendroaspi
L22 0 L21 AND VENOM BLACK MAMBA SNAKE DENDROASPI

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L23 13 L21 AND EG-VEGF

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L24 9 DUP REMOVE L23 (4 DUPLICATES REMOVED)

=> d l24 1-9 cbib abs

L24 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2005:902707 Document No. 143:246742 Chimeric immunogens employing VEGF and helper T-cell peptide epitopes. Kaumaya, Pravin; Cohn, David (The Ohio State University Research Foundation, USA). PCT Int. Appl. WO 2005076972 A2 20050825, 65 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2005-US3747 20050207. PRIORITY: US 2004-2004/PV542041 20040205.
AB The authors disclose compns. and methods of treating patients with malignancies associated with overexpression of VEGF, particularly ovarian cancer. In one example, the compns. comprise VEGF epitopes and chimeric peptides comprising one or more of said epitopes and a T cell epitope.

L24 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2005:523320 Document No. 143:53487 Treatment of rheumatoid arthritis with hypoxia-inducible factor 1α antagonists. Defranoux, Nadine; Hurez, Vincent Jacques; Michelson, Seth G.; Shoda, Lisl Katharine; Wennerberg, Leif Gustaf (Entelos, Inc., USA). PCT Int. Appl. WO 2005053744 A1 20050616, 72 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-US39484 20041124. PRIORITY: US 2003-525363P 20031126.
AB The invention encompasses a novel method of treating inflammatory disease, such as rheumatoid arthritis, and novel methods of identifying and screening for drugs useful in the treatment of inflammatory diseases and their clin. symptoms. The inventors have made the discovery that the activity of HIF-1α, a transcription regulator known to have an

effect on some cancers, has a significant impact on the pathophysiol. of rheumatoid arthritis. The symptoms of an inflammatory disease, such as rheumatoid arthritis, may be alleviated by administering a compound that inhibits the activity of HIF-1α.

L24 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2004:780858 Document No. 141:273369 Compositions comprising human and mouse Bv8 and EG-VEGF with hematopoietic and immune activity and therapeutic uses for blood diseases and immune disorders. Ferrara, Napoleone; Lecouter, Jennifer (Genentech, Inc., USA). PCT Int. Appl. WO 2004081229 A2 20040923, 161 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-US7622 20040312. PRIORITY: US 2003-454462P 20030312; US 2003-511390P 20031014.

AB The present invention relates to the novel expression and activities of Bv8 and EG-VEGF in hematopoietic stem cells (HSCs), lineage-committed blood progenitor cells, and lymphocytes. In particular, Bv8, EG-VEGF, and their receptors are expressed in bone marrow HSCs, peripheral blood leukocytes (PBLs), as well as many hematol. malignant cell lines. Bv8 and EG-VEGF are capable of promoting colony formation of bone marrow mononuclear cells and spleen-derived progenitor cells, increasing populations of white blood cells, and promoting activation of B lymphocytes and T lymphocytes. BvS8 nucleic acids and polypeptides, EG-VEGF nucleic acids and polypeptides, or combinations thereof can be used in a number of assays and in diagnosis and treatment of conditions associated with hematopoiesis, neutropenias, immunodeficiency disorders, and autoimmune disorders.

L24 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
2004:75976 Document No. 140:139512 EG-VEGF receptor antagonists, therapeutic and diagnostic methods, and antagonist identification test system. Haendler, Bernard; Hess-Stumpp, Holger; Schmidt, Anja (Schering A.-G., Germany). Ger. Offen. DE 10229379 A1 20040129, 13 pp. (German). CODEN: GWXXBX. APPLICATION: DE 2002-10229379 20020626.

AB The invention discloses the pharmaceutical use of inhibitors of EG -VEGF-polypeptide and EG-VEGF-nucleic acid and/or the corresponding receptors for treatment and diagnosis of endometriosis and endometrial carcinoma and for treatment of dysfunctional bleeding. The invention further discloses the use of EG-VEGF analogs to increase fertility rates. The invention also discloses a test system for identification of EG-VEGF receptor antagonists.

L24 ANSWER 5 OF 9 MEDLINE on STN DUPLICATE 1
2004388609. PubMed ID: 15292351. Human endocrine gland-derived vascular endothelial growth factor: expression early in development and in Leydig cell tumors suggests roles in normal and pathological testis angiogenesis. Samson Michel; Peale Franklin V Jr; Frantz Gretchen; Rioux-Leclercq Nathalie; Rajpert-De Meyts Ewa; Ferrara Napoleone. (Department of Molecular Oncology, Genentech, Inc., South San Francisco, California 94080, USA.) The Journal of clinical endocrinology and metabolism, (2004 Aug) Vol. 89, No. 8, pp. 4078-88. Journal code: 0375362. ISSN: 0021-972X. Pub. country: United States. Language: English.

AB Angiogenesis is essential for tumor growth and metastasis. A new human angiogenic mitogen, endocrine gland-derived vascular endothelial growth factor (EG-VEGF), has been recently identified; its expression pattern is restricted to endocrine glands, with the highest expression in testis. We used in situ hybridization and newly generated

monoclonal antibodies to investigate the expression of EG-VEGF in normal human prenatal and adult testis and in 48 human testicular tumors of different subtypes. We found that EG-VEGF was expressed from 14 wk until birth in human fetal testis. In the adult testis, EG-VEGF was strongly expressed only in Leydig cells. In testicular tumors, EG-VEGF was expressed specifically in Leydig cell tumors, whereas germ cell-derived neoplasms, including carcinoma in situ, seminoma, and nonseminomatous germ cell tumors, were negative for this antigen. In contrast, VEGF, another powerful angiogenic factor, was expressed in seminoma, but very weakly in Leydig cell tumors. Interestingly, we found that Leydig cell tumors presented vessel surface density 3.2-fold higher than seminoma. These findings argue that human EG-VEGF may play a role in angiogenesis both during the early endocrine development of testis and in the adult testis as well as in Leydig cell tumor growth.

L24 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2003:633928 Document No. 139:175555 Drug screening for inhibitors of peptide ligands for G-protein-coupled receptors ZAQ and 15E as angiogenesis inhibitors. Ohtaki, Tetsuya; Masuda, Yasushi; Takatsu, Yoshihiro (Takeda Chemical Industries, Ltd., Japan). PCT Int. Appl. WO 2003066860 A1 20030814, 308 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (Japanese). CODEN: PIXXD2. APPLICATION: WO 2003-JP1057 20030203. PRIORITY: JP 2002-27299 20020204.

AB Provided are a method and kit for screening compds. inhibiting the activity of novel peptide ligands for two orphan G-protein-coupled receptors ZAQ and 15E. Such compds., antisense nucleic acids or antibodies, are usable as, for example, angiogenesis inhibitors in diagnosis, prevention, and therapy for cancer, polycystic ovary syndrome, ovary overstimulation, etc. The amino acid sequences of those peptides, human, mouse, rat, and bovine ZAQ ligand peptide, snake venom MITI and human and other mammalian homolog (Bv8 peptide), are provided. Endocrine gland-derived vascular endothelial growth factor (EG-VEGF, identical to prokineticin 1) is a novel peptide recently identified as a selective mitogen for endocrine gland endothelial cells. The present study demonstrates that EG-VEGF/prokineticin 1 and a peptide closely related to EG-VEGF, prokineticin 2, are cognate ligands of two orphan G-protein-coupled receptors designated ZAQ (= EG-VEGF/PK-R1) and 15E (= EG-VEGF/PK-R2). EG-VEGF/prokineticin 1 and prokineticin 2 induced a transient increase in intracellular calcium ion concentration ([Ca2+]i) with nanomolar potency in Chinese hamster ovary (CHO) cells expressing EG-VEGF/PK-R1 and -R2 and bind to these cells with high affinity and with different receptor selectivity. EG-VEGF/prokineticins provoke rapid phosphorylation of p44/42 MAP kinase and DNA synthesis in the bovine adrenal capillary endothelial cells (BACE). The mRNAs of both EG-VEGF/PK-R1 and -R2 were expressed in BACE. The identification of the receptors for EG-VEGF/prokineticins may provide a novel mol. basis for the regulation of angiogenesis in endocrine glands.

L24 ANSWER 7 OF 9 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

2003:439594 The Genuine Article (R) Number: 682FM. Differential expression of the angiogenic factor genes vascular endothelial growth factor (VEGF) and endocrine gland-derived VEGF in normal and polycystic human ovaries. Ferrara N (Reprint); Frantz G; LeCouter J; Dillard-Telm L; Pham T;

Draksharapu A; Giordano T; Peale F. Genentech Inc, Dept Mol Oncol, 1 DNA Way, San Francisco, CA 94080 USA (Reprint); Genentech Inc, Dept Mol Oncol, San Francisco, CA 94080 USA; Genentech Inc, Dept Pathol, San Francisco, CA 94080 USA; Univ Michigan, Dept Pathol, Ann Arbor, MI 48109 USA. AMERICAN JOURNAL OF PATHOLOGY (JUN 2003) Vol. 162, No. 6, pp. 1881-1893. ISSN: 0002-9440. Publisher: AMER SOC INVESTIGATIVE PATHOLOGY, INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3993 USA. Language: English. *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Angiogenesis is a key aspect of the dynamic changes occurring during the normal ovarian cycle. Hyperplasia and hypervascularity of the ovarian theca interna and stroma are also prominent features of the polycystic ovary syndrome (PCOS), a leading cause of infertility. Compelling evidence indicated that vascular endothelial growth factor (VEGF) is a key mediator of the cyclical corpus luteum angiogenesis. However, the nature of the factor(s) that mediate angiogenesis in PCOS is less clearly understood. Endocrine gland-derived (EG)-VEGF has been recently identified as an endothelial cell mitogen with selectivity for the endothelium of steroidogenic glands and is expressed in normal human ovaries. in the present study, we compared the expression of EG-VEGF and VEGF mRNA in a series of 13 human PCOS and 13 normal ovary specimens by in situ hybridization. EG-VEGF expression in normal ovaries is dynamic and generally complementary to VEGF expression in both follicles and corpora lutea. A particularly high expression of EG-VEGF was detected in the Leydig-like hilus cells found in the highly vascularized ovarian hilus. in PCOS ovaries, we found strong expression of EG-VEGF mRNA in theca interna and stroma in most of the specimens examined, thus spatially related to the new blood vessels. In contrast, VEGF mRNA expression was most consistently associated with the granulosa cell layer and sometimes the theca, but rarely with the stroma. These findings indicate that both EG-VEGF and VEGF are expressed in PCOS ovaries, but in different cell types at different stages of differentiation, thus suggesting complementary functions for the two factors in angiogenesis and possibly cyst formation.

L24 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

2002:10528 Document No. 136:65270 Protein and cDNA sequences encoding human EG-VEGF protein and methods of use. Ferrara, Napoleone; Watanabe, Colin; Wood, William I. (Genentech, Inc., USA). PCT Int. Appl. WO 2002000711 A2 20020103, 133 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US20116 20010622. PRIORITY: US 2000-213637P 20000623; US 2000-230978P 20000907; WO 2000-US32678 20001201.

AB The present invention is directed to novel polypeptides designated herein as EG-VEGF of human and to nucleic acid mols. encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Also provided herein are methods of screening for modulators of EG-VEGF. Furthermore, methods and related methods of treatment are described herein which pertain to regulating cellular proliferation and chemotaxis.

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2002:964996 Document No. 138:33697 Endocrine gland-derived vascular endothelial growth factor nucleic acids and polypeptides and their

biological activities and use in drug screening and therapies. Ferrara, Napoleone; Watanabe, Colin; Wood, William I.; Shek, Theresa (USA). U.S. Pat. Appl. Publ. US 2002192634 A1 20021219, 105 pp., Cont.-in-part of U.S. Ser. No. 886,242. (English). CODEN: USXXCO. APPLICATION: US 2001-27603 20011219. PRIORITY: US 1998-96146P 19980811; WO 1999-US12252 19990602; US 1999-145698P 19990726; US 1999-380137 19990825; WO 2000-US219 20000105; WO 2000-US4914 20000224; WO 2000-US8439 20000330; US 2000-213637P 20000623; US 2000-230978P 20000907; US 2000-709238 20001108; WO 2000-US32678 20001201; US 2001-886242 20010620.

AB The present invention is based on the identification and characterization of a novel, tissue-restricted, growth and differentiation factor that acts selectively on one endothelial cell type. This factor, referred to as endocrine gland-derived vascular endothelial growth factor (EG-VEGF), induces proliferation, migration, and fenestrations in capillary endothelial cells derived from endocrine glands, but has no effect on a variety of other endothelial and non-endothelial cell types tested. EG-VEGF also induces phosphorylation of kinases involved in cell proliferation or survival, including ERK1, ERK2, Akt, and eNOS. EG-VEGF nucleic acids and polypeptides can be used in a number of assays and in diagnosis and treatment of conditions associated with hormone-producing tissue. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Also provided herein are methods of screening for modulators of EG-VEGF. Furthermore, methods and related methods of treatment are described herein which pertain to regulating cellular proliferation and chemotaxis.

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=> s PTA-4119
L25           0 PTA-4119

=> s "PTA-4119"
L26           0 "PTA-4119"

=> s "PTA-4120"
L27           0 "PTA-4120"

=> s hybridoma
L28       73219 HYBRIDOMA

=> s l28 and PTA-4121
L29           0 L28 AND PTA-4121

=> s l28 and PTA-4122
L30           0 L28 AND PTA-4122

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---Logging off of STN---


=>
Executing the logoff script...


=> LOG Y

COST IN U.S. DOLLARS                        SINCE FILE      TOTAL
                                               ENTRY    SESSION
FULL ESTIMATED COST                           225.32     225.53
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